THE BLOOD PRESSURE IN ARTERIOSCLEROSIS.

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It has long been known that in cases of thickened arteries the blood pressure is likely to be raised. This increased pressure used to be recognized only by the fingers of the examiner, and was referred to as the "tension between the beats." During the last few years, however, many instruments have been introduced for accurately measuring this pressure. Most of these measure not the tension between the beats at all, but the force of the pulse-wave, or, as it is termed, the "systolic pressure." A few, such as that of Erlanger, gauge the pressure between the beats, or, as it is called, the "diastolic pressure;" but most work has been done upon the systolic pressure, which is practically the force of the left ventricle. P. M. Dawson[1] has shown conclusively that the best measure of the systolic output of the heart is the pulse pressure, that is, the difference between the diastolic pressure and the systolic pressure. A patient may have an abnormally high systolic pressure and yet have a dangerously low pressure between the beats, as was the case in a woman examined recently who was dying of aortic regurgitation and yet her systolic pressure was 170 mm. Hg.; her still abnormally strong heart was pumping blood into a comparatively empty arterial tree.

My work has been mostly done with the Martin and the Janeway sphygmomanometers, both of which are modifications of the Riva-Rocci instrument. The former only registers the systolic pressure; the latter is supposed to give also the diastolic pressure, but in my hands at least is very unsatisfactory in this respect. I venture to express the belief that it is the diastolic pressures that we most require to know, and I think that there is great need of a portable instrument that will meet this want.

Although the sphygmomanometer is often of the greatest value in making accurate measurements of blood pressure, the importance of estimating the blood pressure—both systolic and diastolic—by the trained finger is as great as ever, and in many cases is all that is necessary. In the majority of cases, with a little practice, one can even come very near to the systolic pressure in millimeters of mercury by the trained touch alone. The cases in which one is most likely to err are those of very high pressure; it is very difficult, for example, to measure the pressure of a pulse above 200 mm. In arteriosclerosis the first rise that may occur is in the pressure between the beats, and the left heart, as a consequence, becomes stronger than normal, and its amount of hypertrophy is a good gauge of the degree of pressure that it has had to work against.

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[1] Brit. Med. Jour., October 20, 1906.

At one time increased arterial pressure was spoken of as a constant accompaniment of arteriosclerosis, but now that actual measurements can be made, it is found that frequently this pressure is normal or even below normal in such cases. Dunin[2] measured the blood pressure 1000 times in 440 patients with arteriosclerosis in his private practice. He used Gaertner's tonometer in his work. In 120 out of the 440 arteriosclerotic people (27.5 per cent.) the pressure was normal or low. Groedel,[3] of Nauheim, recently published measurements in 500 cases of well-marked arteriosclerosis. In 35 per cent. he found no increase in pressure. Many other workers have come to the same conclusion, and as far as one's small experience warrants, the conclusion is the correct one. Last winter Messrs. A. Ellis and L. B. Robertson, senior medical students in the University of Toronto, investigated this point for me in the General and St. Michael's Hospitals, and their results show that only in about half of the well-marked cases of arterial thickening examined did the pressure run above normal. Occasionally we find a low pressure, when, from the enlargement of the left heart, etc., it is evident that at last the heart is giving up its struggle against long-continued, high pressure. Such a case as the following illustrates this point: A tall, thin man came complaining of great dyspnœa and other signs of cardiac insufficiency. His vessels were markedly thickened and tortuous, and the cardiac apex was felt in the sixth left intercostal space outside the nipple line, and yet the pulse could scarcely be distinguished at the wrist. He was near his end.

Granting that there are many cases of well-marked arteriosclerosis in which the blood pressure is at or below normal, and apparently has never been otherwise, how can we account for the apparent discrepancy? It must be remembered that it takes a great deal of interference with the arterial tree before there is any alteration in the general blood pressure. One may, for example, ligate a large artery, such as the femoral or the common carotid in the dog, without producing any long-continued rise in the general pressure. It is probable that in many of the cases of well-marked sclerosis of the radials the pressure is not raised because the rest of the arterial tract is normal. Localized sclerosis of the splanchnic vessels, however, is said more easily to give rise to increased arterial pressure than is the same amount of disease elsewhere, these vessels being intimately connected with the regulation of the general blood pressure. Hasenfeld and Hirsch, according to Janeway,[4] have definitely proved that arteriosclerosis only leads to increased blood pressure and hypertrophy of the left ventricle when the splanchnic vessels or the aorta above the diaphragm are highly diseased.

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[2] Ztschr. f. klin. Med., liv, Nr. 4.

[3] T. Clifford Allbutt, Brit. Med. Jour., October 20, 1906.

[4] The Clinical Study of Blood Pressure, p. 143.

During some experimental work on blood pressure done some time ago in the Physiological Department of the University of Toronto[5] I came to the conclusion that, while the splanchnic vessels are all-important in the maintaining of blood pressure, yet the vessels below that point are also of great value in the same way; and I should much like to hear of a case of marked sclerosis of the arterial tract below the origin of the splanchnic vessels, and with these vessels normal, in which there was a normal arterial pressure.

When, in a case of arteriosclerosis, we find the pressure to be high, it is necessary to find out how much of this hyperpyesis is due to the disease of the vessel walls and how much to other causes. Careful and repeated examinations of the urine will help toward finding if the condition is due in whole or in part to disease of the kidneys. There may be, and frequently is, a vasomotor element in the case due to toxic or nervous influences, and the free use of the nitrites will assist in gauging the amount of this factor present, the pressure falling much more in such cases than when the pressure is due solely to sclerosis of the vessel walls. Sir Clifford Allbutt[6] writes that "it is a remarkable experience that in cases of great involutionary deformity of the vessels, or, at any rate, of those that are palpable, intercurrent attacks of hyperpyesis may be detected, estimated, and dissipated with as much ease as in persons not so affected."

The question suggests itself here of how much of the normal pressure is dependent upon the activity of the suprarenal capsules. If these are normally active, other influences being undisturbed, the arterial pressure will remain normal. If, however, the suprarenals be sluggish, then the pressure should remain low, and, on the other hand, if they be overactive it should be raised. We know of variations in the functional activity of so many other glands of the body that the conclusion seems almost inevitable that in many cases of disturbed blood pressure we are dealing with examples of over- or under-activity of the suprarenal glands. This interesting question was very ably discussed by Dr. H. D. Rolleston[7] at the last meeting of the Canadian Medical Association, at Montreal.

On the whole, one may say that the finding of a heightened blood pressure is of some value in the diagnosis of a widespread arteriosclerosis, but that, on the other hand, a considerable and often very serious degree of the disease may exist with a normal pressure. I have studied many cases of the latter class in the last few years. One such case much impressed me recently. It was that of a professional man, aged forty-six years, who, after an abstemious but strenuous life, began at the age of forty-three years to suffer from angina pectoris. His right radial was markedly sclerosed, but not so the left or either brachial artery. The blood pressure, upon repeated examination between the attacks, never ranged above 145 mm. The urine was normal. He died in an anginous attack, and the post-mortem examination revealed very marked sclerosis of the coronary arteries, but little disease of the rest of the arterial tree. Another case comes to mind—that of a clergyman, aged sixty-three years, who died of slow cerebral thrombosis. His grandfather had died of apoplexy at seventy years, and the father at sixty-seven years of some similar condition. In this case the arteries open to palpation were not sclerosed beyond what is normal at sixty-three years and the blood pressure was not raised. He had lived a most abstemious life, except as regards "high thinking."

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[5] Observations on Blood Pressure, Univ. of Toronto Study, No. 3.

[6] Loc. cit.

[7] Canada Lancet, November, 1907.

The rise in pressure in cases of sclerosis is usually attributed to the resistance to the onward flow of blood produced by the sclerosed arterial walls. But it is in some cases due possibly to quite a different mechanism. Kocher and Cushing showed some time ago that in localized anemic conditions of vital parts of the brain the blood pressure rises enormously, this rise being evidently an endeavor upon the part of the body to force blood into the dangerously blanched areas. Cook and Briggs[8] also illustrate this well by a chart showing the blood pressure in a case of hemorrhage upon the surface of the brain. The pressure was 360 mm., and after the removal of the clot it fell and remained at normal. Recently a somewhat similar case came under observation: A lady, who had been well except for frequent headaches and some slight hesitation in speech, became for the third time this winter rather suddenly unconscious with signs of acute cerebral compression. During the attack her pressure rose from 125 to 210 mm. Next day, although still profoundly unconscious, with fixed pupils and Babinski's sign in both feet and other signs of marked pressure, her blood pressure was found to be 135 mm. This sign helped one to surmise that the cerebral compression was lessening, and within twelve hours she was quite conscious and is now practically well. These are illustrations of acute compensatory rises in blood pressure, but it seems altogether likely that if the circulation in vital parts of the brain is interfered with by arterial sclerosis, the general blood pressure will rise in the same way in order to get blood into the needful parts.

With the general treatment of arteriosclerosis one is not here concerned, but it may not be out of place to mention a few points from the blood pressure point of view. It is in what has been called the pre-sclerotic stage of the disease that most may be done to relieve the condition. One would fully endorse Sir Clifford Allbutt's sound advice that every adult of say forty-five years and over should, at intervals of two or three years, have his blood pressure taken, in the hope that it may guide his physician to use every endeavor to postpone or prevent the onset of arteriosclerosis. Not that an increased pressure will inevitably produce sclerosis, although if continued long enough and be high, it may do so, but increased pressure and damage to the vessel walls may both be due to some toxin that can, by careful living and treatment, be lessened. In such cases, if we adopt measures such as restricted diet, increased elimination, and the use of the iodides, we may keep the pressure down and are probably at the same time lessening the toxemia which will soon lead to irritation and consequent sclerosis of the vessel walls. It is not advisable in every case of arteriosclerosis immediately to resort to strong measures, such as the nitrites, in order to lower an abnormally high blood pressure. The tissues supplied by the thickened arteries, and it may be vital tissues, are only able to get enough blood for their uses when the pressure is abnormally high.

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[8] Clinical Observations on Blood Pressure, Johns Hopkins Hospital Reports, 1903, vol. xi.

It is wonderful what apparently good health many of these arteriosclerotic people enjoy, even when their pressures are very high. Such a case as the following is of every-day occurrence: A lady, aged fifty-eight years, feels well, except for some breathlessness on exertion and an occasional sense of oppression in the chest. The urine is normal and the arteries moderately sclerosed. Her systolic pressure is constantly between 220 and 235 mm. She has an occasional numbness in the left arm and frequently is dizzy, but does not suffer from headaches. In my experience headaches are not common in arteriosclerotic cases in which the urine remains normal, and I note with interest that G. L. Walton and W. E. Paul[9] recently found, in an analysis of 100 cases of arteriosclerosis, that headache was only present in 22 per cent.

Arteriosclerotic patients often feel weak and shaky and, with the idea of keeping up their strength, take more food than they would use in health. Such a patient came to me about three years ago. Her blood pressure was 240 mm. She was nervous, depressed, and suffered from some headache, and, with the wrong idea that the condition was due to want of nourishment, was taking three good meals a day with lots of meat and eggs, and between meals frequent cups of strong beef tea and sips of whisky. Under a restricted diet, with free saline laxatives and small doses of iodide of potassium, her pressure came down to 190 mm., and she felt much better and the desire for extra food left her. She kept thus fairly well until last month, when she had a hemiplegic attack, and then the urine for the first time was found to be albuminous and the blood pressure over 200 mm.

The onset of early arterial sclerosis should be specially looked for and as far as possible prevented in those individuals whose family history shows that they have a tendency to the disease. Personally, I have seen more cases of the disease in those who have lived abstemious lives than in those who have indulged too freely in the pleasures of the table. In many such cases the only fault that can be found is the strain and stress of modern life acting upon individuals in whom, as Osler graphically puts it, bad material was used in the make-up of the vascular machine.

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[9] Jour. Amer. Med. Assoc., January 18, 1908.

CONCLUSIONS. 1. In many cases of even well-marked arteriosclerosis the blood pressure is not raised. This may be because the disease is localized to a part of the arterial tree, or because the heart may at last be giving up the struggle and hence the pressure, once high, has now fallen.

2. In many cases of arteriosclerosis the blood pressure is above normal.

3. In a given case of arteriosclerosis, in which the pressure is found to be raised, it cannot be assumed that the pressure is high because of the disease of the vessel walls, nor yet that the sclerosis is due to the increased pressure. For example, the arterial stiffening may be due to syphilis, and the hyperpyesis to some intercurrent condition of nervous or toxic nature which may be more or less removable by appropriate treatment.

SYMPTOMS ATTRIBUTED TO THE MYOCARDIUM IN REALITY OFTEN OF PERIPHERAL ORIGIN.

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THE progress of medical thought has always been one of differentiation whereby a group of symptoms which at the time stands for a supposed entity is after a while divided to represent two different conditions, and these are again subdivided, until finally, in place of one disease with a very complex group of symptoms, there is a number of simpler ones with fewer symptoms. Thus, typhus fever once covered typhoid fever, what is now known as typhus fever, and a number of other febrile conditions which have since been more definitely placed. Rheumatism also has been differentiated into a good many conditions which now present no difficulties of distinction.

The same thing is about to happen to that group of heart conditions which has been called myocarditis, corresponding to a predicated organic disease of the heart muscle, which was supposed to produce certain symptoms. It has been customary to attribute many sudden deaths to this condition, while, in fact, it is not logical to believe that a disease characterized by destruction of muscular tissue should terminate suddenly without previous manifestation of muscular weakness. I should be willing once for all to rule out